Chapter 3

Models of single-species populations

3.1 Introduction

It would be easy to follow one example of intraspecific competition with another; each would, in some way, be different from the rest. However, science progresses by not merely accumulating facts but by discovering patterns. We must, therefore, try to pin-point those features of the organism and its environment which are common to many, or even all, of the special cases. We can then concentrate on these essential features in our attempts to understand the dynamics of populations *in general*. We may even be able to discover how best to control the distribution and abundance of organisms, or how to predict the responses of populations to proposed or envisaged alterations in their environment. But in order to do this—or even to make a start in doing this—we require a general conceptual framework on which each special case can be fitted. We require a system that embodies everything that the special cases have in common, but which, by manipulation of its parts, can be made to mirror each of the special cases in turn. In short, we require a model.

As Levins (1968) has suggested, our perfect model would be maximally general, maximally realistic, maximally precise and maximally simple. However, in practice, an increase in one of these characteristics leads to decreases in the others. Each model is, therefore, an imperfect compromise, and it is in this light that models of population dynamics should be seen. Mostly they sacrifice precision and a certain amount of realism, so as to retain a high degree of generality and simplicity. They are usually in the form of an equation or equations illustrated by graphs, but may, occasionally, be in the form of graphs without accompanying equations.

To be useful, and therefore successful, our model of single-species population dynamics should be:
1 a satisfactory description of the diverse systems of the natural and experimental worlds;
2 an aid to enlightenment on aspects of population dynamics which had previously been unclear; and
3 a system which can be easily incorporated into more complex models of interspecific interactions.

We will begin by imagining a population with discrete generations, i.e. one in which breeding occurs in particular seasons only. Having developed a model for this situation, we can return to populations with continuous breeding to develop an analogous model in a similar fashion.

3.2 Populations breeding at discrete intervals

3.2.1 The basic equations

Suppose that each individuals in one generation gives rise to two individuals in the next. If we begin with 10 individuals in the first generation, then the series of population sizes in succeeding generations will obviously be: 20, 40, 80, 160 and so on. This factor by which population size is multiplied each generation is commonly called the *reproductive-rate*, and we can denote it by R, i.e. in the above example $R = 2$.

Note immediately that we are assuming that a *single* figure can characterize reproduction for a *whole* (presumably heterogeneous) population on *all* occasions. Note also, that since 'reproduction' is equivalent to 'birth minus death', we have avoided dealing with birth and death separately. R is, therefore, a '*net* rate of increase' or 'net reproductive-rate'.

We can denote the initial population size (10 in our example) by N_0, meaning the population size when no time has elapsed. Similarly when one generation has elapsed the population size is N_1, when two generations have elapsed it is N_2 and, generally, when t generations have elapsed the population size is N_t. Thus:

Population Ecology: A Unified Study of Animals and Plants Michael Begon, Martin Mortimer and David J. Thompson
 ISBN: 978-0-632-03478-9

$N_0 = 10$,

$N_1 = 20$,

that is:

$N_1 = N_0 \times R$

and, for instance:

$N_3 = N_2 \times R = N_0 \times R \times R \times R = N_0 R^3 = 80$.

In general terms:

$N_{t+1} = N_t R$ (a *difference* equation) (3.1)

and

$N_t = N_0 R^t$ (3.2)

It is plain to see, however, that these equations lead to populations which continue to increase in size indefinitely. The next, obvious step, therefore, is to make the net reproductive-rate subject to intraspecific competition. To do this we will incorporate intraspecific competition into the difference equation.

Consider Fig. 3.1. the justification for point A is as follows: when the population size (N_t) is very, very small (virtually zero), there is little or no competition

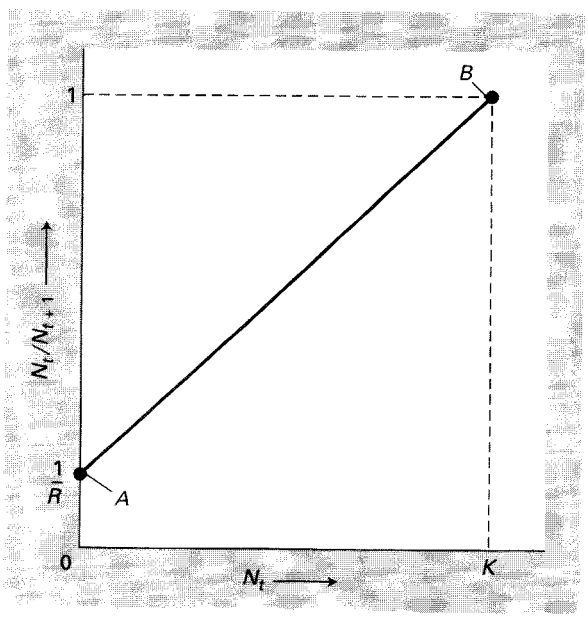


Fig. 3.1 The inverse of generation increase (N_t/N_{t+1}) rising with density (N_t). For further discussion, see text.

in the population, and the net reproductive-rate (R) does not require modification. It is still true, therefore, that $N_{t+1} = N_t R$, or, rearranging the equation:

$$\frac{N_t}{N_{t+1}} = \frac{1}{R}.$$

As population size increases, however, there is more and more competition, and the actual net reproductive-rate is increasingly modified by it. There must presumably come a point at which competition is so great, and net reproductive-rate so modified, that the population can do no better than replace itself each generation. In other words, N_{t+1} is merely the same as N_t (no greater), and N_t/N_{t+1} equals 1. The population size at which this occurs, as we have already seen, is the *carrying-capacity* of the population, denoted by K. This is the justification for point B in Fig. 3.1.

It is clear, then, that as population size increases from point A to point B the value of N_t/N_{t+1} must also rise. But it is for simplicity's sake, and only for simplicity's sake, that we assume this rise follows the straight line in Fig. 3.1. This is because all straight lines are of the simple form: $y = (\text{slope})\,x + (\text{intercept})$. The value for the 'intercept' is clearly $1/R$. That for the 'slope', considering the portion between points A and B, is $(1 - 1/R)K$. The equation of our straight line is, therefore:

$$\frac{N_t}{N_{t+1}} = \frac{\left(1 - \frac{1}{R}\right)N_t}{K} + \frac{1}{R},$$

which, by simple rearrangement, gives:

$$N_{t+1} = \frac{N_t R}{1 + \frac{(R-1)N_t}{K}}.$$

It is probably simpler to replace $(R - 1)/K$ by a and remember:

$$N_{t+1} = \frac{N_t R}{1 + aN_t}. \tag{3.3}$$

The *new* reproductive-rate (repacing the unrealistically constant R) is, therefore:

$$\frac{R}{(1 + aN_t)}.$$

We can now examine the properties of this equation and the net reproductive-rate within it, to see if it is satisfactory: and in some respects it certainly is. Figure 3.2 shows a population increasing in size from very low numbers under the influence of equation 3.3. In the first place, when numbers *are* very low, the population increases in the same, 'exponential' fashion as a population unaffected by competition (equation 3.1). In other words, when:

N_t approaches 0,
$1 + aN_t$ approaches 1,

and

$\frac{R}{1 + aN_t}$ approaches R.

During this initial phase, therefore, the rate at which the population increases in size is dependent only on the size of the population and the potential net reproductive-rate (R) of the individuals within it.

The larger N_t becomes, however, (and thus the more competition there is) the larger $1 + aN_t$ becomes, and the smaller the actual net reproductive-rate $[R/(1 + aN_t)]$ becomes. This situation, in which the net reproductive-rate is under density-dependent control, is responsible for the 'S-shaped' or *sigmoidal* nature of the curve shown in Fig. 3.2. It is important to note, however, that while such a sigmoidal increase is desirable, in as much as its shows our population gradually approaching the carrying-capacity (K) first mentioned in Chapter 2, equation 3.3 is only one of many that would lead to such a pattern. We chose equation 3.3 because of its simplicity.

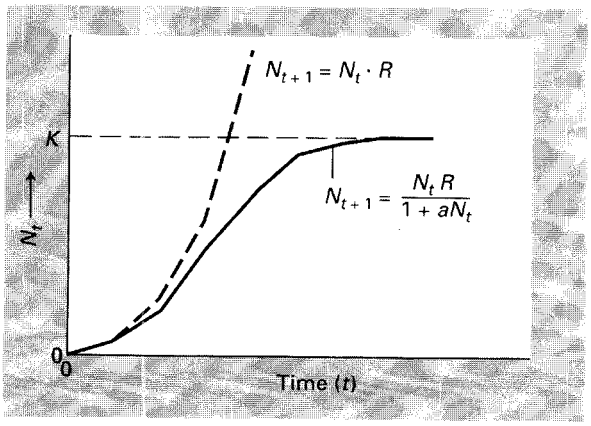


Fig. 3.2 Exponential (dashed) and sigmoidal increase in density (N_t) with time, in a population with discrete generations.

At the top of the curve, N_t approaches K, so that:

$$1 + aN_t \equiv 1 + \frac{(R-1)N_t}{K} \simeq 1 + \frac{(R-1)K}{K} = 1 + (R-1) = R.$$

Thus,

$$N_{t+1} = \frac{N_t R}{1 + aN_t} = \frac{N_t R}{R} = N_t$$

and

$N_t = K = N_{t+1} = N_{t+2} = N_{t+3}$, etc.

If, however, the population is perturbed such that N_t *exceeds* K, then:

$$1 + \frac{(R-1)N_t}{K} > R.$$

and

$N_{t+1} < N_t$.

In other words, the population will return to K. It will also do so if perturbed to below K. The carrying-capacity is a stable equilibrium point to which populations return after perturbation. Our model, therefore, exhibits the regulatory properties classically characteristic of intraspecific competition.

3.2.2 Incorporation of a range of competition

It is clear, from the type of population behaviour that it leads to, that equation 3.3 can describe situations in which a population with discrete generations reproduces at a density-dependent rate. What is not clear is the exact nature of the density-dependence, or the exact type of competition that is being assumed. This can now be examined.

Each generation, the *potential* production of offspring is obviously $N_t R$, i.e. the number of offspring that *would* be produced if competition did not intervene. The *actual* production (i.e. the number that survive) is $N_t R/(1 + aN_t)$. We already know, however,

that the 'k-value' of any population process is given by:

$k = \log_{10}$ (no. produced) $- \log_{10}$ (no. surviving).

In the case of our equation, therefore:

$$k = \log_{10} N_t R - \log_{10}\{N_t R/(1 + aN_t)\},$$

or

$$k = \log_{10} N_t + \log_{10} R - \{\log_{10} N_t + \log_{10} R - \log_{10}(1 + aN_t)\},$$

or

$$k = \log_{10}(1 + aN_t).$$

We also know, from Chapter 2, that the type of intraspecific competition can be determined from a graph of k against $\log_{10} N_t$. Figure 3.3 shows a series of such plots for a variety of values for a and N_0. In each case the slope of the graph approaches 1. In other words, in each case the density-dependence begins by undercompensating, and approaches exact compensation at higher values of N_t. Our model, therefore, lacks the generality we would wish it to have. But such generality is easily incorporated. All we require is a model that can give us, potentially, b-values (slopes of the k versus $\log_{10} N_t$ graph) varying from zero to infinity.

One such general model (and there are others), was originally proposed by Hassell (1975). It is a simple modification of equation 3.3:

$$N_{t+1} = \frac{N_t R}{(1 + aN_t)^b}. \tag{3.4}$$

As Fig. 3.4 indicates, plots of k against $\log_{10} N_t$ will now approach b rather than 1, irrespective of the values of a and N_0. This equation can, therefore, describe populations reacting in a whole range of ways to the effects of competition, and b is the parameter which measures this degree of under- or overcompensation. This whole range is only apparent at higher densities (competition gradually increasing in intensity as N_t increases); but when we remember the actual examples considered in Chapter 2, this seems if anything, a positive advantage of the model. Note, however, that we have still not specified whether it is births or deaths or both which are being affected: to do this would require an even more complex model.

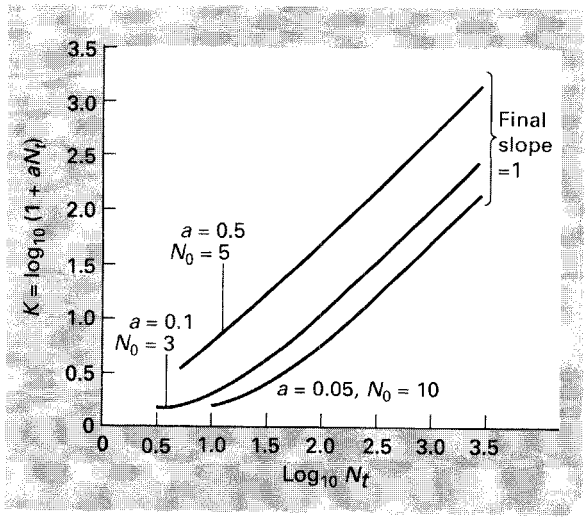


Fig. 3.3 The intraspecific competition inherent in equation 3.3. The final slope (of k against $\log_{10} N_t$) is unity (exact compensation), irrespective of the starting density N_0, or the constant a ($= (R - 1)/K$).

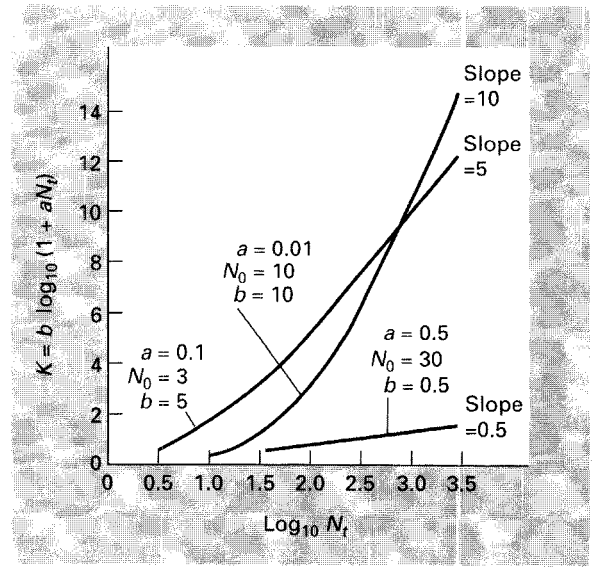


Fig. 3.4 The range of intraspecific competition which can be expressed by equation 3.4. At higher densities, the slope of k against $\log_{10} N_t$ reaches the value of the constant b in the equation, irrespective of N_0 and a.

Nevertheless, provided we remember that we are dealing with a net rate of increase, equation 3.4 can be a useful, simple model.

3.2.3 Models for annual plants

The same model can also be applied to annual plants with discrete generations. A convenient starting point is the law of constant final yield (see Fig. 2.6), the underlying relationship between biomass and density being an inverse one (see Fig. 2.7). Early workers expressed this in a variety of mathematical models (see Willey & Heath, 1969 for a review) but we will concentrate on one recent development. Watkinson (1980) used a generalized expression that can describe both asymptotic and parabolic yield responses to density and also allows a biological interpretation of the constants involved. It expresses average plant size in the populations, $\bar{w}$, as both a function of plant size in the absence of competition w_m and population density, N, after the action of competition (i.e. at harvest);

$$\bar{w} = \frac{w_m}{(1 + aN)^b}. \qquad (3.5)$$

In this, there is clearly more than a passing similarity to equation 3.4. The term $(1 + aN)$ here acts in a number identical to that in equation 3.4, while b allows the incorporation of a range of compensatory responses; w_m, like NR in equation 3.4 is 'what is achieved' in the absence of competition.

To appreciate the features of this model we will explore the hypothetical data presented in Fig. 3.5a, which shows the two general forms of the yield response curve that we have already discussed (the range of units on the axes of the graph are arbitrary). Figure 3.5b shows the corresponding data for mean individual plant size (measured perhaps as biomass) plotted against density. Note initially that the maximum competition-free plant size (w_m) is 100 units of biomass. Generally speaking, the pattern of divergence of the two types of response is determined by the values of the two parameters a and b in equation 3.5. For convenience, however, the value of a ($= 0.1$) is common to both curves. In Fig. 3.5, curves 1 and 2

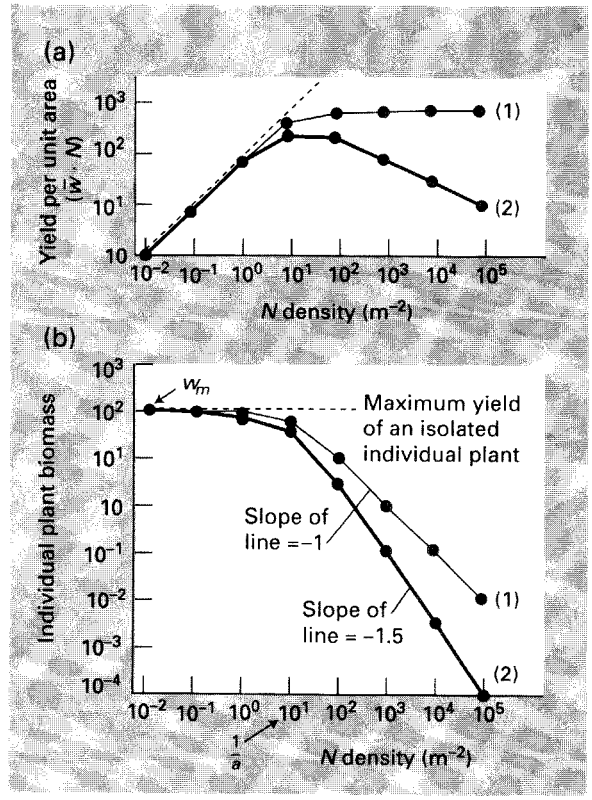


Fig. 3.5 The two general yield-density responses in plant populations expressed on a unit area basis (a) and on an individual plant basis (b), according to equation 3.5. The dotted line indicates a constant proportional relationship between yield and sowing density. (1) Exact compensation, $b = 1$; (2) overcompensation, $b = 1.5$. In both cases, $w_m = 100$ and $a = 0.1$. In (b) the values of the terms in equation 3.5 are indicated for the two yield responses. (The value of 1.5 in curve (2) has no particular significance to the power term of the self-thinning line.) Plant mortality is absent.

both depart appreciably from the density-independent line of constant proportionality (dotted) at $N = 10$ and this in fact is the reciprocal of a in equation 3.5. We can see from Fig. 3.5b that it is at this density that individual plant size begins to be depressed. The reciprocal of this density (i.e. a) is then *broadly* an estimate of the space required for one isolated plant to grow to maximum size. In this arbitrary case, it is clearly 0.1 units of area. This area has been called the

'ecological neighbourhood area' (Antonovics & Levin, 1980; Watkinson, 1981).

Where exact compensation for density is occurring at high density—curve 1 in Fig. 3.5a—plant biomass is directly inversely proportional to density. The slope of the line is -1 (Fig. 3.5b), i.e. $b = 1$. Overcompensation, reflected by values of $b > 1$, results in an enhanced proportional reduction of plant size with density (the slope of the line in Fig. 3.5b is steeper) and yield per unit area is depressed at high density below a maximum occurring at an intermediate one.

The model's most conspicuous and valuable features are that it has the generality to describe the whole range of yield-density responses in the absence of mortality (via the constants a and b) and that the parameters in the model are biologically interpretable.

Equation 3.5, however, does not incorporate mortality of plants. A number of studies have shown that the relationship between density before and after the action of density-dependent mortality follows the graphical form shown in Fig. 3.6. The mathematical description of this is another variant of equations 3.3 and 3.4:

$$N = \frac{N_i}{(1 + mN_i)}. \tag{3.6}$$

Where N_i is the number of plants sown, N is the number surviving and $1/m$ is the maximum population size that can (asymptotically) be reached. We can now combine equations 3.5 and 3.6 to give a model describing the relationship between $\bar{w}$ and N where there are effects both on mortality and growth.

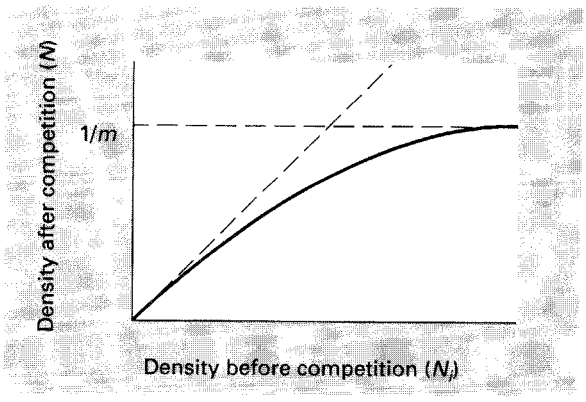


Fig. 3.6 Population sizes before (N_i) and after (N) density-dependent mortality. The curve follows the equation $N = N_i(1 + mN_i)^{-1}$.

In equation 3.5

$$\bar{w} = w_m(1 + aN)^{-b},$$

but N is the number of plants at harvest which is given by equation 3.6. Hence, by substitution,

$$\bar{w} = w_m(1 + aN_i(1 + mN_i)^{-1})^{-b}. \tag{3.7}$$

Furthermore, to calculate yield per unit area at harvest we merely have to multiply $\bar{w}$, (the average plant weight) by N (the density at harvest). We can now examine graphically the behaviour of this model in Fig. 3.7, which shows the yield of a population of plants after density-dependent regulation, in relation to starting population size. In this simulation, as before, each plant when grown under isolated conditions requires 0.1 units of area (a) to achieve a maximum size of 100 biomass units (w_m). Where there is exact compensation at high densities ($b = 1$), the effect of decreasing the maximum population size attainable ($1/m$) is to lower yield. If, however, there is overcompensation ($b > 1$) the shape of the yield-density curve depends on the intensity of density-dependent mortality (governed through m) in relation to the ecological neighbourhood area (a). Overt overcompensation is greatest when the potential maximum population size ($1/m$) is highest. Hence, the model has the generality to encompass the range of density responses which we know to occur. Furthermore, it incorporates the basic mechanisms of plasticity and mortality that are part of the response. We must bear in mind though that it is only a static description of an essentially dynamic process occurring within plant populations.

Up to now we have been concerned only with the relationship between plant size after the action of competition and initial population density (i.e. at sowing), but it is a relatively easy step to extend our model to one that describes changes from generation to generation. To do this we need to know the relationship between plant biomass at harvest, $\bar{w}$, and the number of seeds produced per plant, S. Often this may be described by

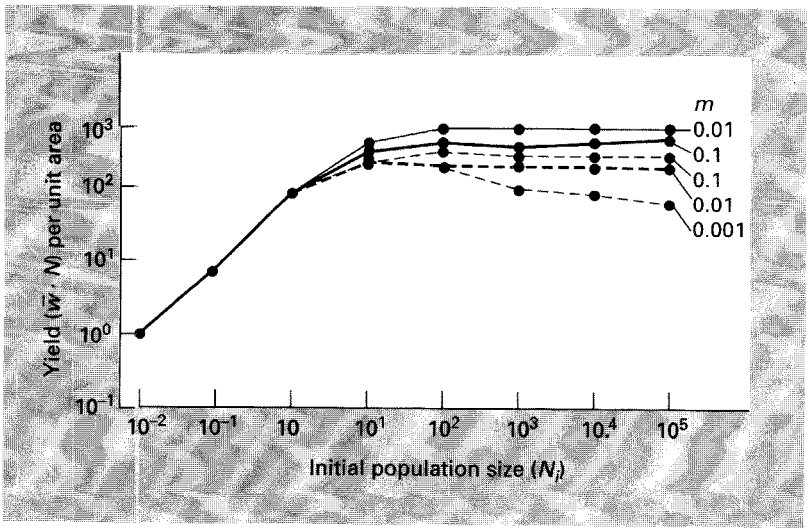


Fig. 3.7 The relationship between initial population size N_i and yield per unit area as determined by equations 3.6 and 3.7. Constants in the model are $a = 0.1$, $w_m = 10$, $b = 1.0$ (solid line), $b = 1.5$ (dotted lines). Values of m as indicated.

$$S = q\bar{w}^p$$

Where q and p are constants describing the exact form of the relationship. We may therefore express seed number per plant at harvest as a function of seeds sown in an identical way to equation 3.7:

$$S = \lambda(1 + aN_i(1 + mN_i)^{-1})^{-b}$$

where λ is the number of seeds produced by a plant in isolated (non-competitive) conditions. Multiplying both sides of the equation by the number of surviving plants gives us the seed yield per unit area, and hence we have a population model relating the number of seeds sown to those harvested. Thus

$$SN = \lambda(1 + aN_i(1 + mN_i)^{-1})^{-b}N.$$

Since N_i and SN are the population sizes in successive generations, we may replace them by N_t and N_{t+1}, remembering that we are dealing with populations of seeds. Upon rearrangement our model becomes

$$N_{t+1} = \frac{\lambda N_t}{(1 + aN_t)^b + m\lambda N_t}.$$

On inspection, this too is very similar to equation 3.4, differing only in so far as it includes an additional term for density-dependent mortality: $m\lambda N_t$. In the absence of mortality ($m = 0$), this model contracts to equation 3.4 and will of course display the properties already discussed.

3.3 Continuous breeding

We can now return to populations which breed continuously. For our purposes, the essential difference between these and ones with discrete generations is that population size itself changes continuously rather than in discrete 'jumps'. This is illustrated in Fig. 3.8.

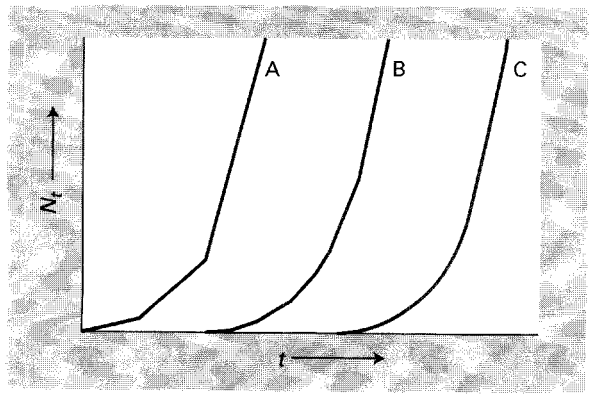


Fig. 3.8 Three populations growing exponentially at the same rate. A has long, discrete generations; B has short, discrete generations; C breeds continuously.

Curves A and B are both of the form $N_{t+1} = N_t R$, and represent populations increasing (exponentially) at essentially the same rate. ('Rate' in this case means 'the amount by which population size (N_t) increases per unit time (t)'.) The difference between them is that generation time is much shorter in curve B than curve A. The same process has been extended in curve C until the straight line segments are so small (infinitesimally small) that the graph is a continuous curve. Nevertheless, the rate at which the population increases has remained essentially the same. Curve C describes a situation in which, during each and every exceedingly small time interval, there is the possibility, at least, of birth and death. In other words, breeding and dying are continuous. Thus, the initial equation we require in order to build our general model of a continuously breeding population must retain the essential properties of $N_t = N_0 R^t$, but must describe a continuous curve.

Differentiation is a mathematical process which is specifically designed to deal with changes occurring during infinitesimally small intervals of time (or of any other measurement on the x-axis). Those familiar with differential calculus will clearly see (and those unfamiliar with it need merely accept) that differentiating $N_t = N_0 R^t$ by t we get:

$$\log_e N_t = \log_e N_0 + t \log_e R,$$

and

$$\frac{dN}{dt} \times \frac{1}{N} = 0 + 1 \log_e R,$$

or

$$\frac{dN}{dt} = N \log_e R.$$

This is the equation we require. dN/dt is the *slope* of the curve, defining the *rate* at which population size increases with time. $\log_e R$ is usually replaced by r, 'the intrinsic rate of natural increase' or 'instantaneous rate of increase', but the change from R to r is simply a change of currency: the commodity dealt with remains the same (essentially 'birth minus death'). The *differential* equation $dN/dt = rN$, just like the difference equation $N_{t+1} = N_t R$, describes a situation of exponential population increase dependent only on the size of the population and the reproductive-rate of individuals.

We can proceed now in Fig. 3.9 exactly as we did in Fig. 3.1. The rate of increase *per individual* is unaffected by competition when N approaches zero, and is, therefore, given by r (point A). When N reaches K, the carrying-capacity, the rate of increase per individual is 0 (point B). As before, we assume that the line between A and B is straight, and thus:

$$\frac{dN}{dt} \times \frac{1}{N} = \frac{-r}{K} \times N + r,$$

or

$$\frac{dN}{dt} \times \frac{1}{N} = r\left(1 - \frac{N}{K}\right),$$

and

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right). \tag{3.8}$$

This is the so-called *logistic equation*. Its characteristics are essentially the same as those of the difference equation 3.3 described previously. Minor dissimilarities, associated with the change from the difference to the differential form, will be discussed later; and, of course, it describes a continuous sigmoidal curve,

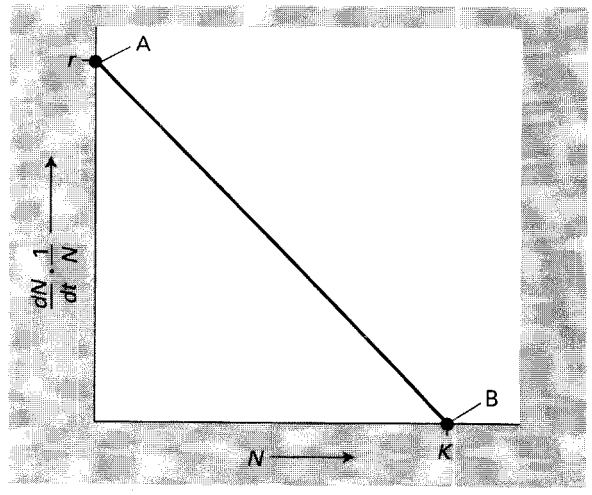


Fig. 3.9 The rate of increase per individual $dN/dt \cdot 1/N$ falling with density (N). For further discussion, see text.

rather than a series of straight lines. Note once again, moreover, that there are many other equations which would lead to sigmoidal increase; the justification of the logistic is the simplicity of its derivation. Note, too, that the logistic equation is based on exact compensation, in just the same way as its discrete generation analogue. In the case of the logistic, however, it is by no means easy to incorporate a factor, such as b, which will generalize the model to cover all types of competition. Our equation for a continuously breeding population must, therefore, remain comparatively imperfect.

Drawing these arguments together then, we are left with two equations: equation 3.4 as an apparently satisfactory general model for discrete-generation increase and equation 3.8 as a somewhat less satisfactory model for the behaviour of a continuously breeding population.

3.4 The utility of the equations

3.4.1 Causes of population fluctuations

We can now proceed to examine the models' utility. The extent to which they can be incorporated into more complex models of interspecific interactions will become apparent in Chapters 4 and 5. We shall begin here by determining whether our models can indeed throw important *new* light on aspects of population dynamics. To do so we shall examine the question of fluctuations in the sizes of natural, single-species populations. That *some* degree of fluctuation in size is shown by *all* natural populations hardly needs stressing.

The causes of these fluctuations can be divided into two groups: extrinsic and intrinsic factors. Amongst the extrinsic factors we include the effects of other species on a population, and the effects of changes in environmental conditions. These are topics which will be discussed in later chapters. For now, we will concern ourselves with intrinsic factors. Our approach will be to examine our models to see which values of the various parameters, and which minor alterations to the models themselves, lead to population fluctuations; and to see what type of fluctuation they lead to.

The method by which our discrete-generation equation may be examined has been set out and discussed by May (1975). It is a method which uses fairly sophisticated mathematical techniques, but we can ignore these and concentrate on May's results. These are summarized in Fig. 3.10. Remember that Fig. 3.10a refers to equation 3.4. It describes the way in which populations fluctuate (Fig. 3.10b) with different values of R and b inserted. (The value of a, i.e. $(R-1)/K$, determines the level about which populations fluctuate, but not the manner in which they do so.) As Fig. 3.10a shows, low values of b and/or R lead to populations which approach their equilibrium size without fluctuating at all. Increases in b and/or R, however, lead firstly to damped oscillations gradually approaching the equilibrium; and then to 'stable limit cycles' in which the population fluctuates around the equilibrium level, revisiting the same two, or four, or even more points time and time again. Finally, with large values of b and R, we have a population fluctuating in a wholly irregular and chaotic fashion.

The *biological* significance of this becomes apparent when we remember that our equation was designed to model a population which *regulated* itself in a density-dependent fashion. We can see, however, that if a population has even a moderate net reproductive-rate (and an individual leaving $100(=R)$ offspring in the next generation in a competition-free environment is not unreasonable), and if it has a density-dependent reaction which even moderately overcompensates, then *far from being stable*, it may fluctuate in numbers without any extrinsic factor acting. Thus, our model system has *taught* us that we need not look beyond the intrinsic dynamics of a species in order to understand the fluctuations in numbers of its natural populations. It is worthwhile stressing this. Our intuition would probably tell us that the ability of a population to regulate its numbers in a density-dependent way should lend stability to its dynamics. Yet our model shows us that if this regulation involves a moderate or large degree of overcompensation, and at least a moderate reproductive-rate, then the population's numbers may fluctuate considerably because of those very 'regulatory' processes. As May, himself, concludes: '...even if the natural world was 100% predictable, the dynamics of populations with "density-

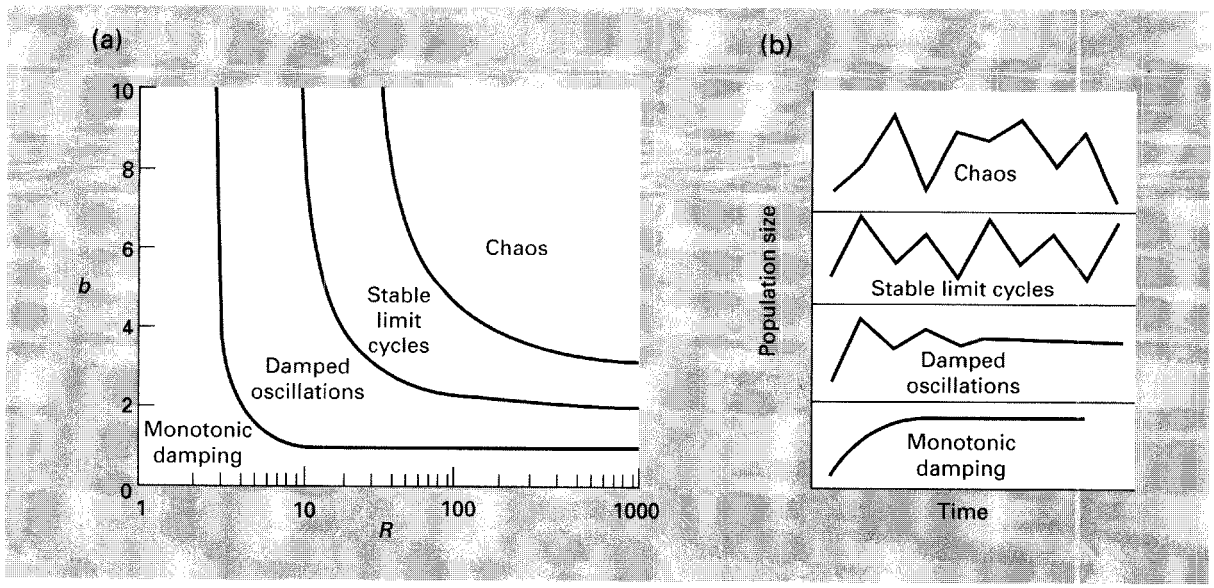


Fig. 3.10 The effect of intraspecific competition on population dynamics. (a) The range of population fluctuations (themselves shown in (b)) generated by equation 3.4 with various combinations of b and R inserted. For further discussion, see text. (After May, 1975.)

dependent" regulation could nonetheless in some circumstances be indistinguishable from chaos' (see also section 6.12).

In examining this general model, the special, exact-compensation case ($b = 1$) has itself been covered. Irrespective of R, the population will reach K without overshooting; and with R-values greater than about 10 this will take little more than a single generation. (The analogous differential equation—the logistic—behaves very similarly to this: always approaching K with exponential damping.) This special case can be modified, however, to incorporate an additional feature. We have assumed until now that populations respond *instantaneously* to changes in their own density. Suppose, on the contrary, that the reproductive-rate is determined by the amount of resource available to the population, but that the amount of resource is determined by the density of the previous generation. This will mean that the reproductive-rate is dependent on the density of the *previous* generation. Thus, since:

$$N_{t+1} = N_t \times \text{reproductive-rate}$$

$$N_{t+1} = \frac{N_t R}{1 + aN_{t-1}}. \tag{3.9}$$

In other words, there is a *time-lag* in the population's response to its own density, caused by a time-lag in the response of its resource. The amount of grass in a field in spring being determined by the level of grazing the previous year is a simple but reasonable example of this, the behaviour of this modified equation, as shown by computer simulations, is as follows:

$R <$ approx. 1.3 : exponential damping

$R >$ approx. 1.3 : damped oscillations.

In comparison, the original equation, without a time-lag, led to exponential damping for all values of R. *The time-lag has provoked the fluctuations.* Once again, therefore, examination of our model has taught us which intrinsic features of a species' dynamics can lead to fluctuations in its population density.

In fact, there are other types of time-lag and they all *tend* to provoke fluctuations in density. Consider again the difference between a population with discrete generations and one with continuous breeding. In both cases, the population responds throughout each

'time interval' to the density at the start of that time interval. With continuous breeding the time intervals are infinitesimally small, and the response of the population is, therefore, continually changing; conversely with discrete generations the population is still responding at the end of a time interval to the density at its start. In the meantime, of course, this density has altered: there is a time-lag. Thus, the difference equation model is more liable to lead to fluctuations than the differential logistic, and it is the time-lag which accounts for the difference. Moreover, some organisms may respond to density at one point in their life cycle, and actually reproduce some time later; this 'developmental time' between response and reproduction is also a time-lag. In all cases, the population is responding to a situation that has already changed, and an increased level of fluctuation is the likely result.

There are two important conclusions to be drawn from this discussion. The first is that time-lags, high reproductive-rates and highly overcompensating density-dependence (either alone or in combination) are capable of provoking all types of fluctuation in population density, without invoking any extrinsic cause. The second conclusion is that this is clear to us only because we have studied the behaviour of our model systems.

3.4.2 The equations as descriptions

Finally, we must examine the ability of our models to describe the behaviour of natural and experimental systems. There are two aspects of this. The first is concerned with the way in which single-species populations increase or fluctuate in size; the second with the precise way in which intraspecific competition affects the various facets of fecundity and survival.

A number of examples of population increase and fluctuation are illustrated in Fig. 3.11. Figure 3.11a describes the continuous increase of a population of yeast cells under laboratory conditions (Pearl, 1927): the resemblance to the logistic curve is quite striking. Figure 3.11b, conversely, describes the year-by-year change in the size of the sheep population of Tasmania (Davidson, 1938). There is no obvious detailed resemblance between this and the pattern predicted by any of our models. Nevertheless, the initial rise in numbers, decelerating as it approaches a plateau (albeit a fluctuating one), is reminiscent of the pattern generated by our difference equation model with b close to 1. If, furthermore, we take into account the fluctuations in environmental conditions, the interactions of the sheep with other species, and the undoubted imperfections of the sampling method, then the resemblance between Fig. 3.11b and our model begins to look more significant. This is the nub of our problem in deciding how satisfactory our models are. On the one hand, the field data, unlike some data collected under controlled conditions, do not follow our models' behaviour *exactly*. On the other hand, there is nothing in the field data to make us *reject* our models. We could claim, in fact, that our models represent a very satisfactory description of the population dynamics of sheep in Tasmania, but that the complexities of the real world tend to blur some of the edges. Similar conclusions could be drawn from Fig. 3.11c–e, although in each case the model concerned might be slightly different; perhaps a different value for a and b, or the addition of some sort of time-lag. Figure 3.11c, for example, would be quite adequately described by our difference equation with $R = 20$ and $b = 2$; the pattern in Fig. 3.11d could be accounted for, simply, by environmental fluctuations; and the fluctuations in numbers of *Daphnia* (Fig. 3.11e) do appear, quite genuinely, to be caused by a time-lag.

The most reasonable conclusion seems to be this. Our models *seem to be* satisfactory, in as much as they are *capable* of describing the observed patterns of increase and fluctuation as long as environmental fluctuations, interspecific interactions and the imperfections of sampling are taken into account. Conversely, the discrepancies between our models and our data could be due, quite simply, to the essential inadequacies of our models, particularly the unrealistic simplifying assumptions they make. To examine their utility more critically, we must consider their ability to describe the effects of intraspecific competition on fecundity and survival. It should also be stressed, however, that a true test of a model's ability

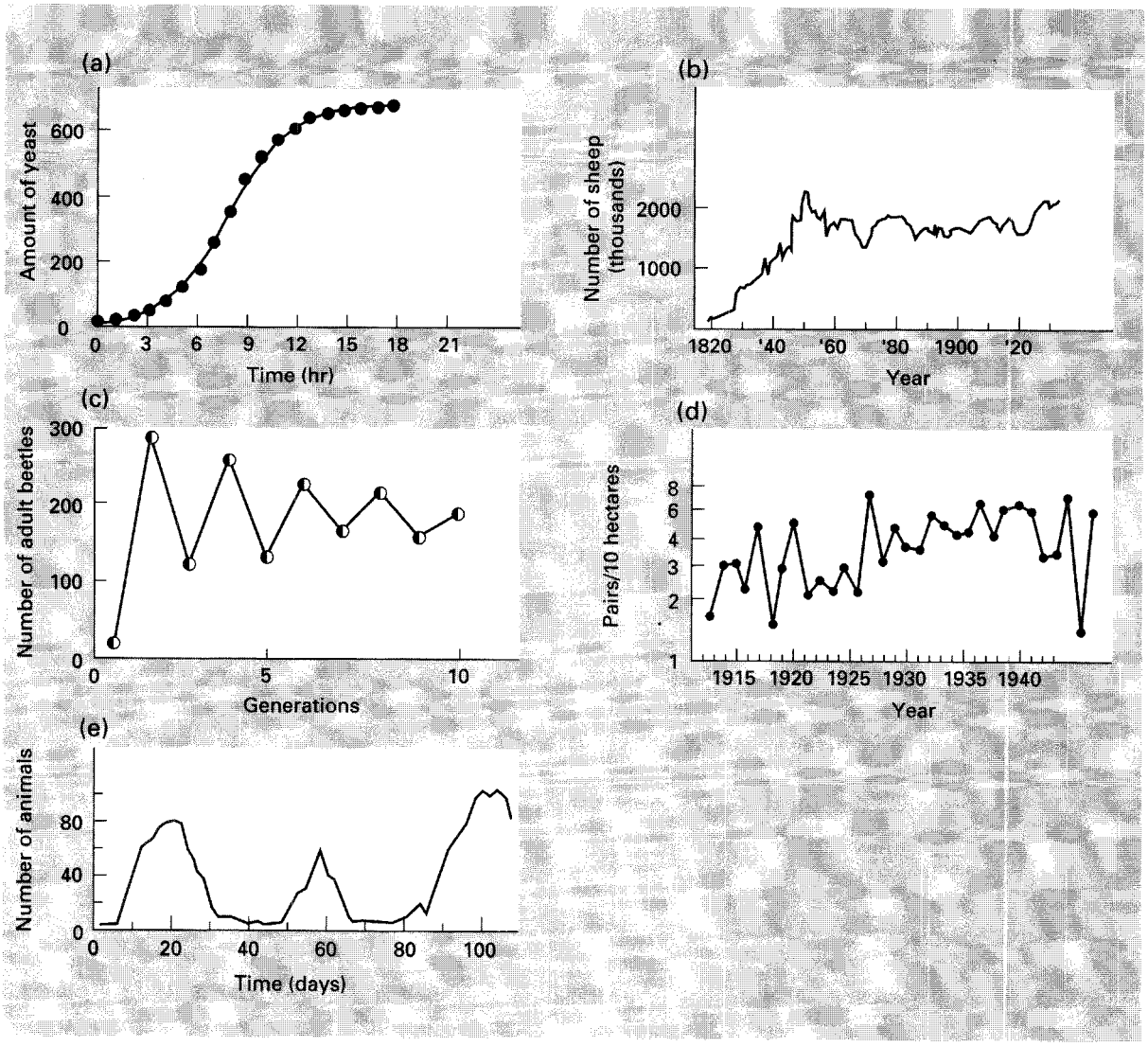


Fig. 3.11 Observed population fluctuations. (a) Yeast cells. (After Pearl, 1927.) (b) Tasmanian sheep. (After Davidson, 1938.) (c) The stored-product beetle *Callosobruchus maculatus*. (After Utida, 1967.) (d) The great tit *Parus major* in Holland. (After Kluyver, 1951.) (e) The water flea *Daphnia magna*. (After Pratt, 1943.)

to describe population behaviour should not consist only of a comparison of *numbers*; it should also consider the underlying *biological* similarities: the respective R-values for instance, or the existence—in both model and fact—of a time-lag.

In considering the ability of our models to describe the effects of intraspecific competition, we must confine ourselves to the discrete-generation equation. The logistic model, restricted as it is to perfect compensation, cannot be expected to describe a range of situations; its utility lies in its simplicity and the ease with which it can be understood. The capabilities of our difference equation can be critically assessed, because, for any set of data the most appropriate values for a and b—those giving the 'best fit' to the data—can be estimated using a statistical technique

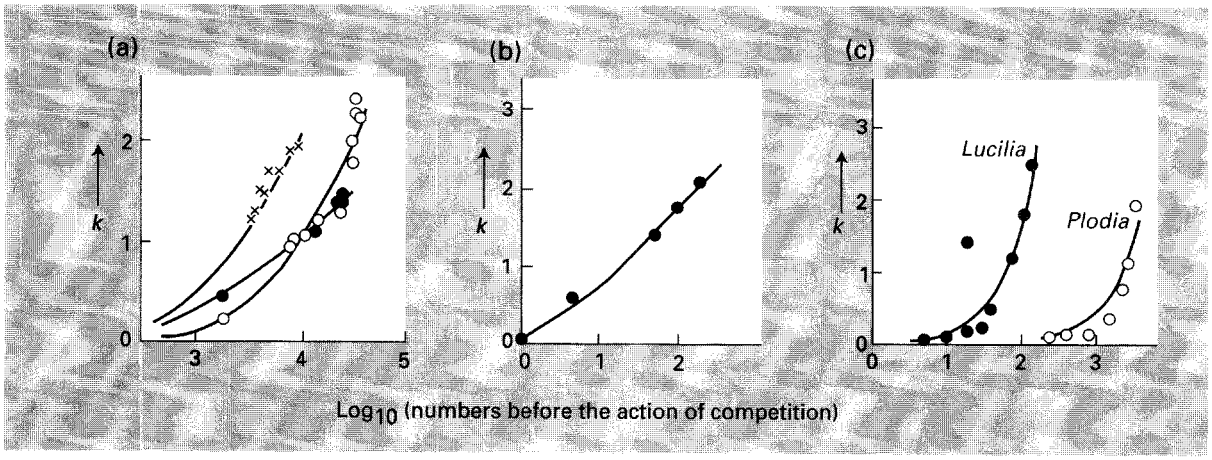


Fig. 3.12 Data from competition experiments and their description based on equation 3.4. (a) Beetles: *Callosobruchus chinensis* (●) $a = 0.00013$, $b = 0.9$ (Fujii, 1968); *C. maculatus* (×) $a = 0.0006$, $b = 2.2$ (Utida, 1967); *C. maculatus* (○) $a = 0.0001$, $b = 2.7$ (Fujii, 1967). (After Hassell *et al.*, 1976.) (b) Shepherd's purse, *Capsella bursa-pastoris*: $a = 0.377$, $b = 1.085$ (Palmblad, 1968). (c) the blowfly *Lucilia cuprina* (Nicholson, 1954b); the moth *Plodia interpunctella* (Snyman, 1949). (After Hassell, 1975.)

(following Hassell, 1975 and Hassell *et al.*, 1976). The curve produced by the model can then be compared with the original data points. This has been done for several examples (including two from Chapter 2), and the results are shown as plots of k-values against $\log_{10}$ density in Fig. 3.12 (experimental data) and Fig. 3.13 (field data). As originally noted by Hassell (1975), there is a tendency, particularly under laboratory conditions, for the model to be very satisfactory at high and low densities, but incapable of describing the sudden transition from a shallow to a steep slope. This may be a result of the data reflecting two superimposed density-dependent processes (one weak and one strong), while the model treats them as a single process (Stubbs, 1977). Nevertheless, the model's performance overall is impressive: whether the plot is more or less straight or curved, its fit to the data is very satisfactory.

A final example of the utility of the difference equation model is shown in Fig. 3.14. The data come from a pot experiment in which *Agrostemma githago*, corncockle, was sown over a wide range of densities. We can see that there were two components of density-dependent regulation: plant mortality and plasticity. Self-thinning was noticeable above sowing densities of 1000 seeds m^{-2} (Fig. 3.14a), whilst the number of seeds borne per flowering plant declined with density over the entire range (Fig. 3.14b).

Fitting the models (equations 3.5 and 3.6) described above to these data by statistical means, we can see that they do indeed give a very close fit to the observed results. It appears (again) that our basic difference equation model is very satisfactory in describing reality. Since, however, we can interpret the constants in the models in biological terms we can make further statements about the *Agrostemma* population. The data suggest that the maximum population size that can be supported (if the experiment is repeated again under identical conditions) is 10 869 seed-bearing plants m^{-2} ($= m^{-1} = 1/(9.2 \times 10^{-5})$), and that the maximum seed yield of an isolated plant, λ, is 3685 seeds. Also, to grow to maximum size, a plant required 325 cm^2 ($a = 0.0325$) of space. The value of b (1.15) in equation 3.6 was significantly greater than unity, indicating overcompensation at high density. This is evident when the components of seed yield are examined (Fig. 3.14c). The effects of density did not fall equally on all parts of the plant. Most density stress was absorbed by the number of capsules per plant which declined at a constant rate with density as did seed weight. The number of seeds per capsule,

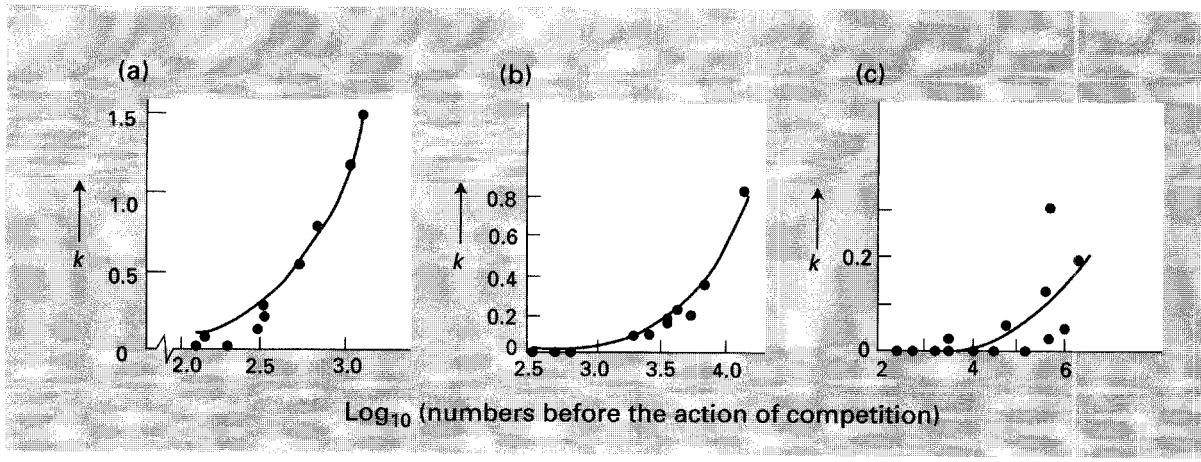


Fig. 3.13 Field data on intraspecific competition and their description based on equation 3 and 4. (a) The limpet *Patella cochlear*: $a = 4.13 \times 10^{-5}$, $b = 51.49$ (Branch, 1975). (b) The Colorado beetle *Leptinotarsa decemlineata*: $a = 3.97 \times 10^{-6}$, $b = 30.95$ (Harcourt, 1971). (c) Larch tortrix *Zeiraphera diniana*: $a = 1.8 \times 10^{-5}$, $b = 0.11$ (Auer, 1968). (After Hassell, 1975.)

however, fell sharply at high density reflecting the overcompensatory density response. The difference equation model, despite its simplicity, does indeed appear to encapsulate the essential characteristics of the behaviour of single-species populations.

3.4.3 'Cobwebbing'—a more general approach

Whilst the behaviour of populations described by models such as equation 3.4 can be examined mathematically, there will of course be situations where we cannot find appropriate functions relating N_{t+1} to N_t and even when we can, they may not be tractable analytically. In such cases, however, we can examine the behaviour of the population by *geometric iteration*, bearing in mind that this method will have its limitations (see below). In the simplest case for discrete generations, the approach involves plotting the size of the population after a generation of growth against its former (or starting) size. In section 1.2 we introduced this idea of examining the dynamics of a population graphically by plotting its size over successive generations, each generation against the next (see Fig. 1.3). One of the earliest uses of this approach was in fisheries research in the 1950s in investigations of sustainable fishing of natural stocks.

If the procedure is carried out to encompass a number of generations, then we might envisage a curve of the form illustrated in Fig. 3.15. The curve has been termed a recruitment or reproduction curve. To investigate the behaviour of the population we examine the changes in population size by comparing the reproduction curve with a one to one reference line ($N_{t+1} = N_t$), plotting the data on log scales. Any population lying on this reference line will be at equilibrium.

Four theoretical recruitment curves are illustrated in Fig. 3.15 and we can predict the size of the population over succeeding generations by iteration using a method known as cobwebbing the reproduction curve (Hoppensteadt, 1982). In the first (Fig. 3.15a) the recruitment curve parallels the reference line and for a chosen starting size (N_t) we may evaluate N_{t+1} on the reproduction curve and reflect this value back to the horizontal axis through the one to one line. Inspection of Fig. 3.15a shows that the population exhibits exponential growth increasing in size by a factor of 10 each generation.

When growth-rate is density-dependent, reproduction curves will not parallel the reference line but will intersect it at a locus which by definition is an equilibrium population size N_e. Of particular interest, however, is the slope of the reproduction curve at the

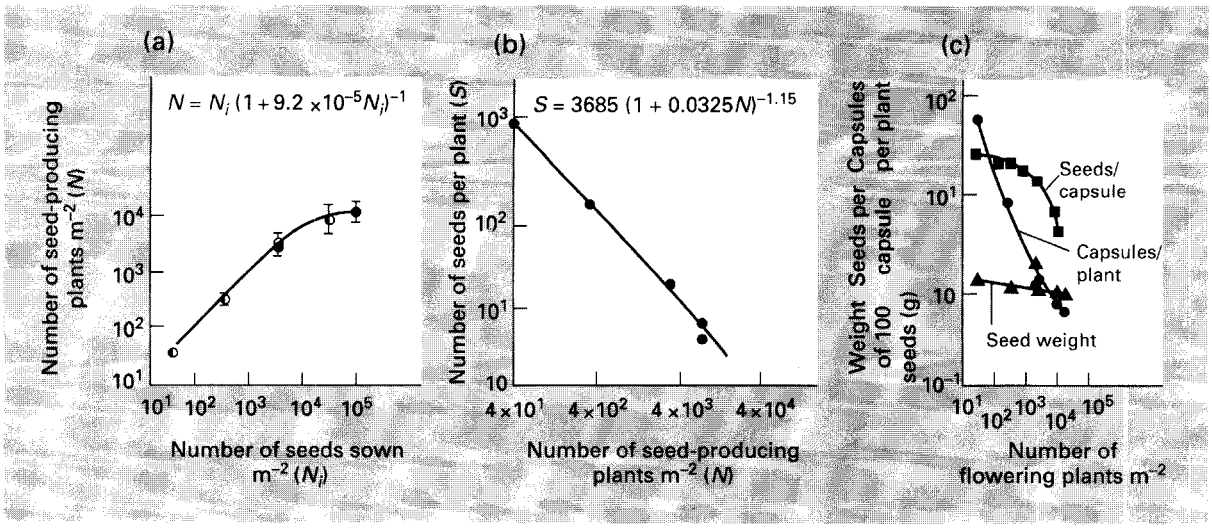


Fig. 3.14 Density-dependent regulation in *Agrostemma githago*: (a) survival to reproduction; (b) seed production; (c) yield components. (From Watkinson, 1981.)

approach to the point of intersection. Where this slope is in the range of zero to +1 (indicating either undercompensating density-dependence (slope > 0) or exactly compensating density-dependence (slope = 0)) we can see that population shows a monotonic approach to the equilibrium point. Where the population approaches the equilibrium point from below, the incremental gain diminishes with each generation, and when it approaches from above, the incremental decline diminishes with each generation (Fig. 3.15b). Population perturbations away from this density in either direction will result in a smooth return to it.

Reproduction curves, however, may exhibit a 'hump' such that population growth rate is maximal at some intermediate density between N_e and very low densities. This is indicative of overcompensating density-dependence. Two theoretical examples are shown in Fig. 3.15c and 3.15d, and by cobwebbing we can illustrate two further forms of behaviour. Where the slope of the reproduction curve is negative but less than −1 in the region of N_e (mildly overcompensating), damped oscillations towards a stable equilibrium population size will occur (Fig. 3.15c). If, however, the slope is greater than −1 (more strongly overcompensating), then as Fig. 3.15d indicates population sizes may fluctuate around N_e and in a chaotic manner—a pattern we have already observed in the circumstances for the specific case of equation 3.4, and to which we will return in section 6.12.

Figure 3.16 provides selected reproduction curves that have been observed in natural or laboratory populations and we will return to use them in explaining mechanisms of population regulation, particularly in plants. Before we leave this topic, we must voice a cautionary word of warning. Whilst intuitive and simple, graphical analysis by cobwebbing is limited since it will not necessarily expose the full range of dynamical behaviour that a population may exhibit (see Yodzis, 1989). For example, in particular circumstances, the outcome will depend on the population size chosen to start the exploration.

3.5 Incorporation of age-specific fecundity and mortality

We have seen from the life-table data for annual meadow grass and red deer (Chapter 2) that mortality and fecundity are often age-specific. The models we have developed so far, however, do not include these important features, and to this extent they are deficient. It may seem a formidable task to attempt to model a population of overlapping generations in

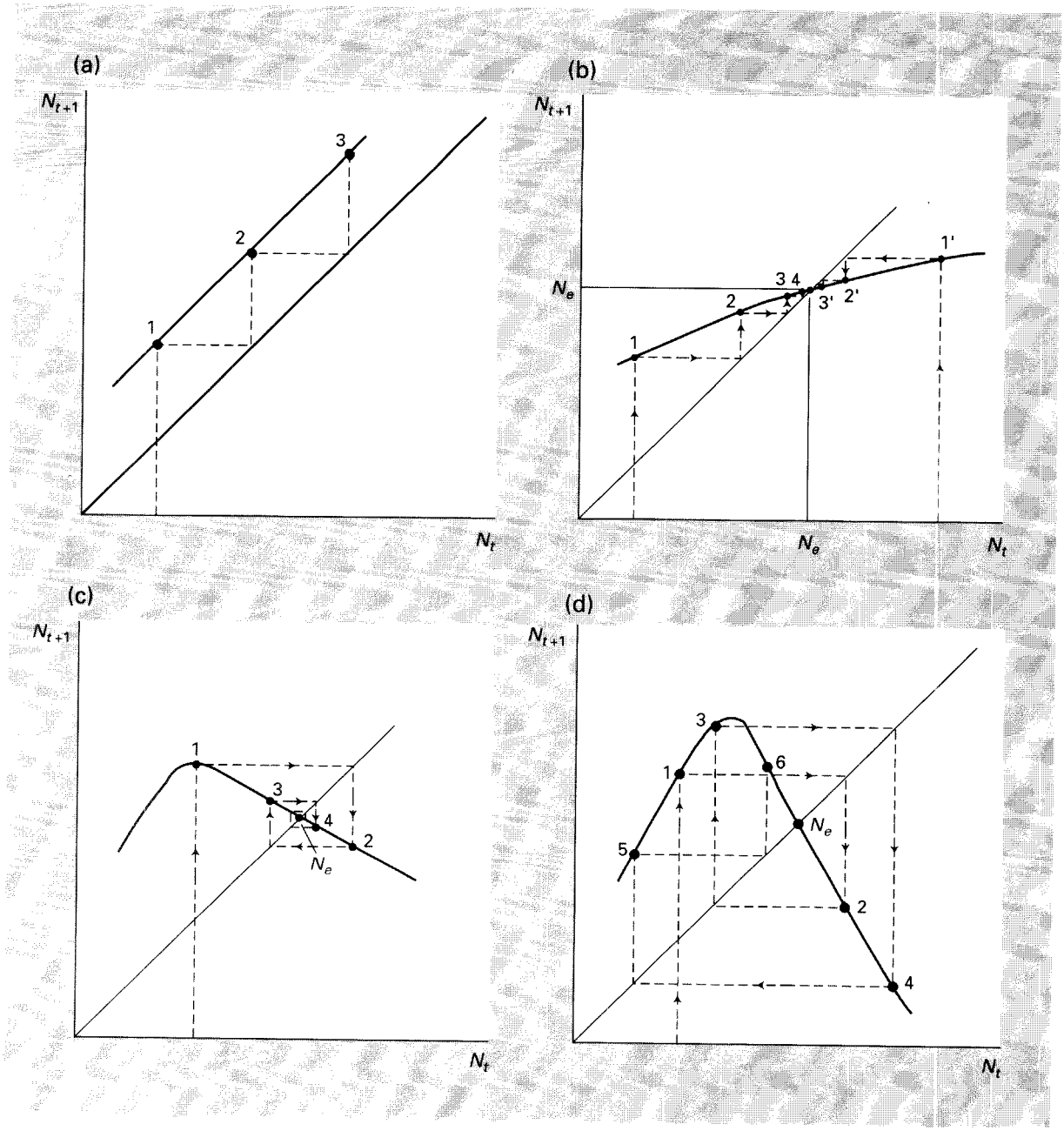
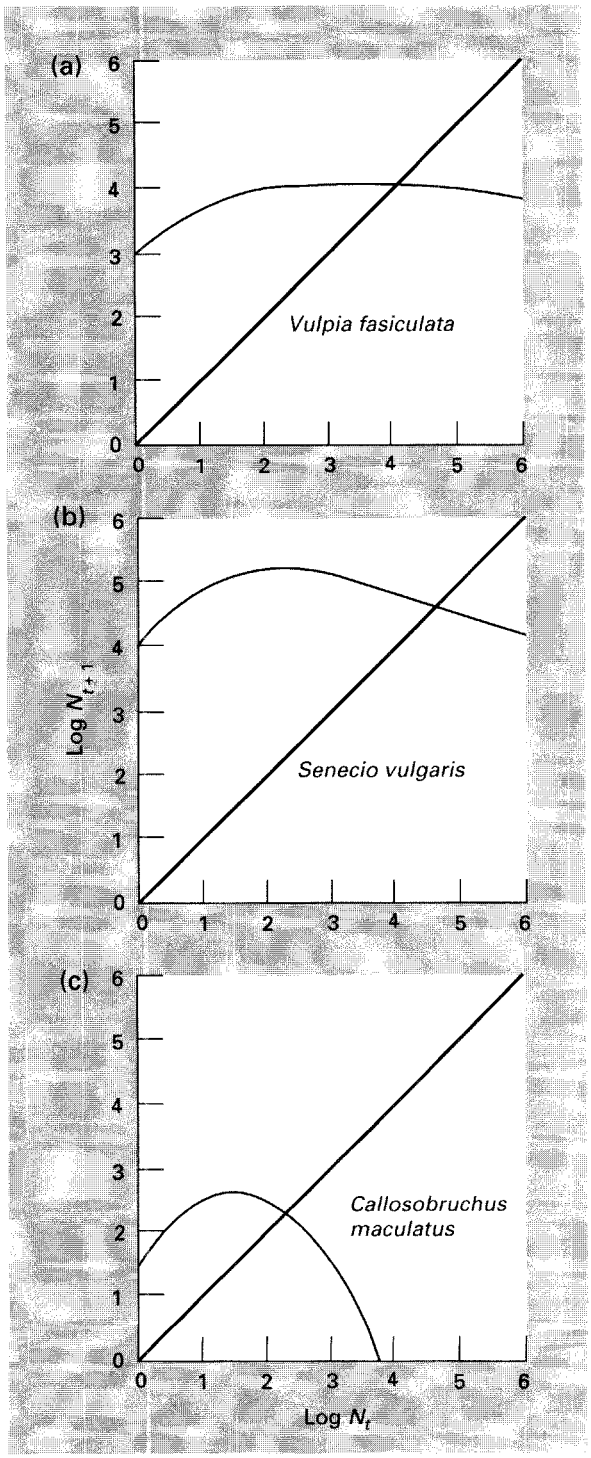


Fig. 3.15 Theoretical recruitment (or reproduction) curves. In each case (a–d) the size of the population after a generation of growth is plotted against initial size. Dotted lines illustrate the process of cobwebbing. (a) Exponential growth; (b) monotonic approach to equilibrium N_e; (c) damped oscillations towards equilibrium; (d) chaotic behaviour (see text for details).

these terms, but by the application of appropriate mathematical techniques it can be achieved fairly easily.

The best starting point is the diagrammatic life table for overlapping generations (see Fig. 1.9), expanded into a more complete form (Fig. 3.17). Here we sup-



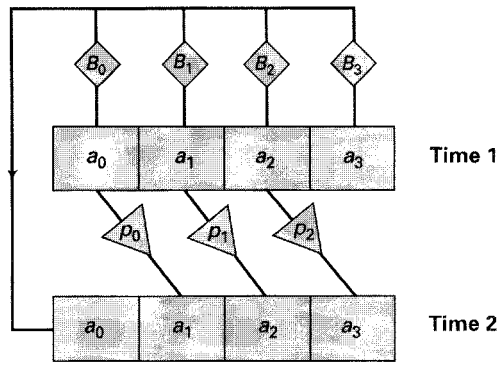


Fig. 3.17 The diagrammatic life table for a population with overlapping generations: a, numbers in different age groups; B, age-specific fecundities; p, age-specific survivorships.

pose that a population can be conveniently divided into four age groups: a_0, a_1, a_2 and a_3; a_0 representing the youngest adults and a_3 the oldest. In a single time step, t_1 to t_2, individuals from group a_0, a_1 and a_2, pass to the next respective age group; each age group contributes new individuals to a_0 (through birth); and the individuals in a_3 die. This clearly rests on the assumption that the population consists of discrete age groupings and has discrete survivorship and birth statistics, in contrast to the reality of a continuously ageing population.

We can write a series of algebraic equations to express the changes that are occurring in Fig. 3.17. These are:

$$ {}_{t_2}a_0 = ({}_{t_1}a_0 \times B_0) + ({}_{t_1}a_1 \times B_1) + ({}_{t_1}a_2 \times B_2) + ({}_{t_1}a_3 \times B_3) \quad (3.10) $$

$$ {}_{t_2}a_1 = ({}_{t_1}a_0 \times p_0) \quad (3.11) $$

$$ {}_{t_2}a_2 = ({}_{t_1}a_1 \times p_1) \quad (3.12) $$

$$ {}_{t_2}a_3 = ({}_{t_1}a_2 \times p_2) \quad (3.13) $$

Fig. 3.16 Representative reproduction curves for (a) *Vulpia fasiculata* (Watkinson & Harper, 1978); (b) *Senecio vulgaris* (Watson, 1987); (c) *Callosobruchus maculatus* (Utida, 1967)—population fluctuations of these data are shown in Fig. 3.11c.

where the numbers in the age group are subscripted t_1 or t_2 to identify the time period to which they refer. There are four equations because there are four age groups, and they specifically state how the numbers in age groups are determined over the time-step t_1 to t_2.

Suppose that our population at t_1 consists of 2000 individuals distributed among the age groups as follows:

$${}_{t_1}a_0 = 1750,\ {}_{t_1}a_1 = 100,\ {}_{t_1}a_2 = 100,\ {}_{t_1}a_3 = 50.$$

Suppose, further, that the age-specific birth- and survival-rates are as follows:

Age	*B*	*p*
0	0	0.1
1	5	0.6
2	15	0.3
3	10	0.0.

Then, for instance, no offspring are born to adults aged a_0, whilst adults in age group a_2 are the most fecund. Also, only 10% of the individuals aged a_0 survive the next time-step and become incorporated into a_1, whereas none of the individuals in a_3 survive ($p = 0$). The numbers at time t_2 will be:

$$\begin{aligned}
{}_{t_2}a_0 &= (1750\times 0)+(100\times 5)+(100\times 15)+(50\times 10) = 2500\\
{}_{t_2}a_1 &= 1750\times 0.1 = 175\\
{}_{t_2}a_2 &= 100\times 0.6 = 60\\
{}_{t_2}a_3 &= 100\times 0.3 = 30\\
&\qquad\qquad 2765
\end{aligned}$$

During this time-step our population has increased by 765 individuals and our age distribution has changed:

from	a_0 =	1750 to	2500
	a_1 =	100	175
	a_2 =	100	60
	a_3 =	50	30

It is important to realize that equations state that *individuals reproduce before they die*. Taking age group a_2, for instance, the figure 100 was used in equation 3.10 to compute the number of births ($100 \times 15 = 1500$), but *then* used in equation 3.13 to compute the number of a_2 survivors into age group a_3 ($100 \times 0.3 = 30$).

3.5.1 The matrix model

The equations 3.10–3.13 are called 'linear recurrence equations', but it is clear that in this form our model is rather cumbersome, consisting as it does of as many equations as there are age groups. One way of expressing our model in a much more compact form is by the use of matrix algebra. In essence, matrix algebra is a tool designed to manipulate and store large sets of data. We will certainly not need to cover all aspects of matrix algebra to understand how our model is constructed in matrix terms, but it is vital that the basic concepts are clearly understood.

A matrix is simply a group, or table, or array of numbers. For instance, the numbers for B and p envisaged above could be set out as the matrix:

$$\begin{bmatrix} 0 & 0.1 \\ 5 & 0.6 \\ 15 & 0.3 \\ 10 & 0.0 \end{bmatrix}$$

and we signify that it is a matrix by surrounding the numbers by square brackets. Conventionally, matrices are symbolized by a letter in bold face, say **X**.

The number of rows and columns that comprise a matrix may vary, and we may have matrices consisting of only a single column. We can, in fact, write the initial age structure of our previous population in this way:

$$\begin{bmatrix} 1750 \\ 100 \\ 100 \\ 50 \end{bmatrix}$$

and symbolize it as ${}_{t_1}\mathbf{A}$. Our age distribution at t_2, ${}_{t_2}\mathbf{A}$, can obviously then be written as

$$\begin{bmatrix} 2500 \\ 175 \\ 60 \\ 30 \end{bmatrix}$$

The two matrices ${}_{t_1}\mathbf{A}$ and ${}_{t_2}\mathbf{A}$ are technically called *column vectors*, indicating that our matrices are in fact just one column of figures.

We have seen how our population changes from ${}_{t_1}\mathbf{A}$

to ${}_{t_2}\mathbf{A}$ through the medium of the recurrence equations. Now, to complete our matrix model, we have to construct a suitable matrix to enable ${}_{t_1}\mathbf{A}$ to become ${}_{t_2}\mathbf{A}$ by mutiplication. The construction of this matrix is determined by the rules of matrix multiplication, as we shall learn below, and in consequence it appears as:

$$\begin{bmatrix} 0 & 5 & 15 & 10 \\ 0.1 & 0 & 0 & 0 \\ 0 & 0.6 & 0 & 0 \\ 0 & 0 & 0.3 & 0 \end{bmatrix} = \mathbf{T}.$$

Note that matrix **T** is square, and that we have entered into it all our age-specific survival and birth statistics in particular positions, writing all the other numbers (or matrix *elements* as they are called) as zero.

The multiplication of our initial, age-distributed population occurs according to the rules of matrix algebra, as follows. Take, in turn, each *row* of elements in **T**. Each individual element in the row is multiplied with the corresponding element in ${}_{t_2}\mathbf{A}$: the first with the first, the second with the second, and so on. These pairwise multiplications are then summed, and the sum entered as the appropriate element in a new column vector. Thus, the first row of the square matrix leads to the first element of the new column vector, the second leads to the second, and so on. This may be illustrated as:

$$\begin{bmatrix} 0 & 5 & 15 & 10 \\ 0.1 & 0 & 0 & 0 \\ 0 & 0.6 & 0 & 0 \\ 0 & 0 & 0.3 & 0 \end{bmatrix} \times \begin{bmatrix} 1750 \\ 100 \\ 100 \\ 50 \end{bmatrix}$$

$$= \begin{bmatrix} (0)(1750)+(5)(100)+(15)(100)+(10)(50) \\ (0.1)(1750)+(0)(100)+(0)(100)+(0)(50) \\ (0)(1750)+(0.6)(100)+(0)(100)+(0)(50) \\ (0)(1750)+(0)(100)+(0.3)(100)+(0)(50) \end{bmatrix} = \begin{bmatrix} 2500 \\ 175 \\ 60 \\ 30 \end{bmatrix}$$

We can observe now that the positions of the zeros in the matrix **T** are critical, because they reduce terms in each row of the multiplication to zero, giving rise in ${}_{t_2}\mathbf{A}$ to the age structure that we have already seen.

This matrix model was first introduced to population biology by P.H. Leslie in 1945 and is often known as the Leslie matrix model. In general form, for n age groups, it is:

$$\begin{bmatrix} B_0 & B_1 & B_2 & \dots & B_{n-1} & B_n \\ p_0 & 0 & 0 & \dots & 0 & 0 \\ 0 & p_1 & 0 & \dots & 0 & 0 \\ 0 & 0 & p_2 & \dots & 0 & 0 \\ \cdot & \cdot & \cdot & & \cdot & \cdot \\ \cdot & \cdot & \cdot & & \cdot & \cdot \\ \cdot & \cdot & \cdot & & \cdot & \cdot \\ 0 & 0 & 0 & \dots & p_{n-1} & 0 \end{bmatrix} \times \begin{bmatrix} {}_{t_1}a_0 \\ {}_{t_1}a_1 \\ {}_{t_1}a_2 \\ {}_{t_1}a_3 \\ \cdot \\ \cdot \\ \cdot \\ {}_{t_1}a_n \end{bmatrix} = \begin{bmatrix} {}_{t_2}a_0 \\ {}_{t_2}a_1 \\ {}_{t_2}a_2 \\ {}_{t_2}a_3 \\ \cdot \\ \cdot \\ \cdot \\ {}_{t_2}a_n \end{bmatrix} \quad (3.14)$$

which may, alternatively, be written:

$$\mathbf{T} \times {}_{t_1}\mathbf{A} = {}_{t_2}\mathbf{A}. \quad (3.15)$$

T is called the *transition* matrix, which, when *post-multiplied* by the vector of ages at t_1, gives the age distribution at t_2. (In matrix algebra, in contrast to conventional algebra, $\mathbf{T} \times \mathbf{A}$ is different from $\mathbf{A} \times \mathbf{T}$ (cf. $x \times b = b \times x$), and we distinguish between the two by referring to post- and pre-multiplication.)

By now it must be clear that our matrix model allows us to condense the complexities of age-specific schedules into a simply written but explicit form. It might also seem that to use the model requires endlessly repeated multiplications and additions. This disadvantage has been removed, however, by the widespread use of computers, which are ideally suited (not to say designed) to perform such iterative procedures at speed. Our model is, therefore, of great value.

The specific rules and procedures of matrix algebra are numerous. We have only dealt here with those which are necessary in this particular context. If further explanations are needed, reference may be made to one of the several books on matrix algebra for biologists (e.g. Searle, 1966).

3.5.2 Using the model

If we now wish to compute the changing size of our population, on the assumption that the birth and mortality statistics are constant from one time to the next, we can write:

$$\mathbf{T} \times {}_{t_1}\mathbf{A} = {}_{t_2}\mathbf{A}; \qquad \mathbf{T} \times {}_{t_2}\mathbf{A} = {}_{t_3}\mathbf{A}; \qquad \mathbf{T} \times {}_{t_3}\mathbf{A} = {}_{t_4}\mathbf{A},$$

and so on. This is a process of iterative pre-multiplication of the successive age groups by the

transition matrix, giving:

$$T \times \begin{bmatrix} 1750 \\ 100 \\ 100 \\ 50 \end{bmatrix} = \begin{bmatrix} 2500 \\ 175 \\ 60 \\ 30 \end{bmatrix}; \quad T \times \begin{bmatrix} 2500 \\ 175 \\ 60 \\ 30 \end{bmatrix} = \begin{bmatrix} 2075 \\ 250 \\ 105 \\ 18 \end{bmatrix};$$

and

$$T \times \begin{bmatrix} 2075 \\ 250 \\ 105 \\ 18 \end{bmatrix} = \begin{bmatrix} 3005 \\ 207 \\ 150 \\ 32 \end{bmatrix}$$

Over these three time-steps the age distribution is changing and the population is increasing in size; the numbers of individuals in all age-groups are oscillating. If we were to continue the iteration, however, these oscillations would disappear (Fig. 3.18), and after 17 and 18 multiplications the vectors would be

$$\begin{bmatrix} 24845 \\ 2110 \\ 1070 \\ 272 \end{bmatrix} \text{ and } \begin{bmatrix} 29320 \\ 2484 \\ 1266 \\ 321 \end{bmatrix}$$

respectively. Note that the population has grown enormously, but that the ratios of ${}_{t_{18}}a_x : {}_{t_{17}}a_x$ are all equal (to two significant figures at least), i.e.

$$\frac{29320}{24845} = \frac{2484}{2110} = \frac{1266}{1070} = \frac{321}{272} = 1.18.$$

This means that we have reached a situation in which the age distribution is proportionally constant or *stable* as the population increases. In other words, $a_0 : a_1 : a_2 : a_3$, is the same in all subsequent generations. In fact, this particular stable age distribution is an attribute of the transition matrix, and would have been reached irrespective of the initial column vector; and in general, populations, when subjected to repeated pre-multiplication by the same transition matrix, achieve the stable age distribution characteristic of that matrix.

Moreover, as Fig. 3.18 shows, the ratios from one generation to the next (1.18 in this case) are also constant: the population (as well as each of the age-classes within it) is increasing at a constant rate. This rate is also a reflection of the transition matrix, and is,

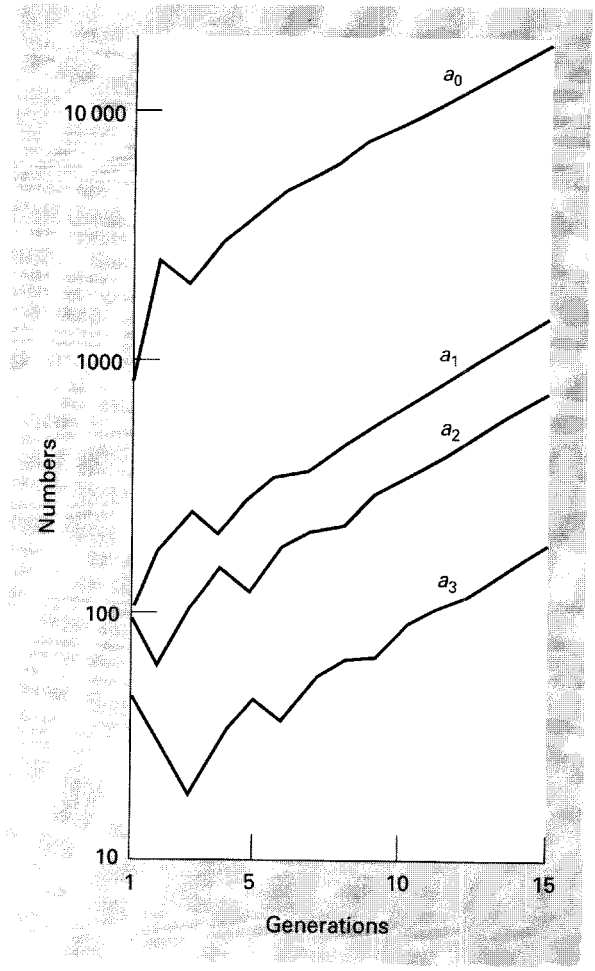


Fig. 3.18 The behaviour of a matrix population model: oscillations in an age-distributed population as a stable age distribution is approached. See text for details.

in fact, the net reproductive-rate or finite rate of increase per unit of time, R. Repeated pre-multiplication is, therefore, the mathematically simplest means of determining R from an age-specific schedule of births and deaths.

We should not, however, simply dismiss the matrix model as a device for only handling sets of recurrence equations in an iterative manner. A transition matrix, stating age-specific data as it does, is a precise description of the population statistics and can be subjected to algebraic analysis. First, we can find R, by calculating 'the dominant latent root' of the

transition matrix; and second, we can conduct sensitivity analyses to investigate the relative importance of the different transitions in determining population growth-rate. Both techniques require a knowledge of matrix algebra which is beyond the scope of this book (but which are clearly discussed in Caswell, 1989). A further usage of transition matrices comes in harvesting theory (Chapter 5), which allows us to predict the level of 'cropping' that can occur without driving a population to extinction. All that need be appreciated at this stage is that matrix modelling can lead to sophisticated analyses of populations and their behaviour.

Our computations of R and the stable age distribution rest on the assumption that the elements of the transition matrix are *constant over time* and *independent of population density*. In these respects our matrix model, in its present form, is very much divorced from the biological reality of natural populations. We can incorporate temporal changes in fecundity and survival in a very straightforward manner, by simply changing our transition matrix with every time-step. Thus, whereas we have assumed that:

$$\mathbf{T} \times {}_{t_1}\mathbf{A} = {}_{t_2}\mathbf{A} \text{ and } \mathbf{T} \times {}_{t_2}\mathbf{A} = {}_{t_3}\mathbf{A},$$

so that

$$\mathbf{T} \times \mathbf{T} \times {}_{t_1}\mathbf{A} = {}_{t_3}\mathbf{A},$$

we can assume instead that, in general terms, for r time periods:

$$\mathbf{T}_r \times \mathbf{T}_{r-1} \times \mathbf{T}_{r-2} \ldots \mathbf{T}_2 \times \mathbf{T}_1 \times {}_{t_1}\mathbf{A} = {}_{t_r}\mathbf{A} \qquad (3.16)$$

Furthermore, we can incorporate the idea that the birth and death statistics are density-dependent by varying the fecundity and survival elements in the transition matrix in relation to population size. To achieve this, we need simply derive the relevant elements of the successive transition matrices from equations which relate fecundity and survivorship to the population size, which is itself the sum of the elements of the most recent age vector.

3.5.3 A working example: *Poa annua*

Both of these additions to the model are well illustrated in practice by the work of Law (1975) on the annual meadow grass *Poa annua*: a particularly successful colonizer of open habitats, which responds strongly to intraspecific competition. In modelling populations of this species, Law envisaged that the life cycle included four ages of plants besides seeds (Fig. 3.19), and that the span of each age group was approximately 8 weeks. The transition matrix appropriate to this life cycle is given in Table 3.1 and, although most of the elements are familiar to us, the incorporation of a seed bank into the model requires

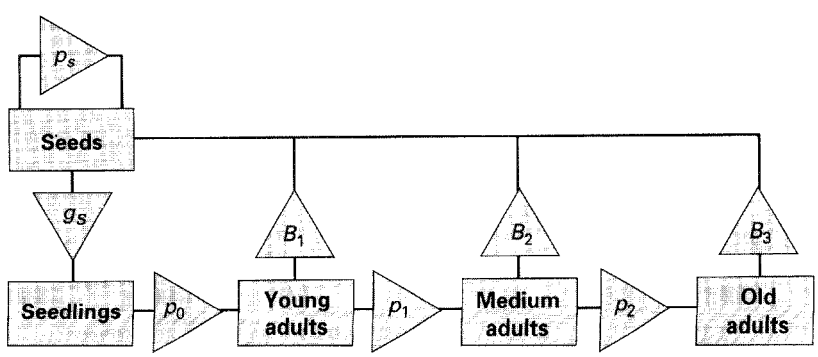


Fig. 3.19 The life cycle for annual meadow grass *Poa annua*. (After Law, 1975.) Proportions (ranging from 0 to 1): p_s, seeds surviving; g_s, seeds germinating; p_0, seedlings surviving to become young adults; p_1, young adults surviving to become medium adults; p_2, medium adults surviving to become old adults. Fecundities (0 or >0): B_1, B_2 and B_3, seed produced by young, medium and old adults, respectively. All events occur over the time period t to $t + 1$.

Table 3.1 A matrix model of the life cycle outlined in Fig. 3.19.

$$\begin{bmatrix} p_s & 0 & B_1 & B_2 & B_3 \\ g_s & 0 & 0 & 0 & 0 \\ 0 & p_0 & 0 & 0 & 0 \\ 0 & 0 & p_1 & 0 & 0 \\ 0 & 0 & 0 & p_2 & 0 \end{bmatrix}$$

Table 3.2 A transition matrix, **P**, for a *Poa annua* population. (After Law, 1975).

$$\begin{bmatrix} 0.2 & 0 & B_1(N) & B_2(N) & B_3(N) \\ 0.05 & 0 & 0 & 0 & 0 \\ 0 & p_0(N) & 0 & 0 & 0 \\ 0 & 0 & 0.75 & 0 & 0 \\ 0 & 0 & 0 & 0.75 & 0 \end{bmatrix}$$

that the first element of the matrix (p_s) is not a fecundity but the probability of a seed surviving in the bank if it does not germinate.

Law was able to develop curves of both seedling survivorship to young adults, and age-specific seed output per plant in relation to overall population density. These are shown in Fig. 3.20 together with the equations that describe these relationships. The transition matrix which incorporates these density-dependent functions of survival and reproduction takes the form shown in Table 3.2. Between successive time intervals, 0.2 of the seeds in the banks remain dormant and survive, while 0.05 of them germinate. The proportion of individuals that survive from the seedling age-class, $p_0(N)$, is initially 0.75 at very low population density, but declines according to the function shown in Fig. 3.20. The proportion of individuals that survive from the young adult age-class onward, however, is fixed at 0.75. This represents the specific assertion that the survivorships of individuals in these age-classes are not subject to density-dependence. The maximum seed production of young and old adults at very low population density is 100 seeds per plant, but, with increasing density, this decreases in a negative exponential fashion. Similarly, the maximum seed production of medium adults is 200 seeds per plant, which also decreases with density. This reflects *P. annua*'s schedule of fecundity in which seed output peaks in the medium adult age-class (see Fig. 1.13).

The matrix model,

$$\mathbf{P} \times {}_{t}\mathbf{A} = {}_{t+1}\mathbf{A}$$

can now be used to simulate the behaviour of the population, which is depicted in Fig. 3.21. We can see that each group of individuals undergoes oscillations in numbers, but when 18 time periods have elapsed the size of each has stabilized and population increase

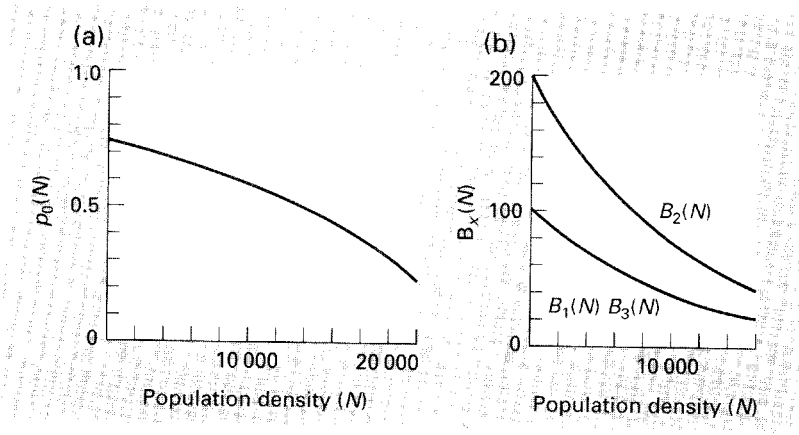


Fig. 3.20 (a) The proportion of seedlings of *Poa annua* surviving to become young adults as a function of population density: $p_0(N) = 0.75 - 0.25 \exp(0.00005\ N)$ if $N < 27726$; $p_0(N) = 0$ if $N > 27726$. (b) Age-specific seed production per plant for young (B_1) medium (B_2) and old (B_3) adults of *Poa annua* as a function of population density: $B_1(N) = B_3(N) = 100 \exp(-0.0001\ N)$; $B_2(N) = 200 \exp(-0.0001\ N)$. (From Law, 1975.)